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Acute coronary artery dissection after multiple bee stings

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ABSTRACT

The occurrence of an acute coronary syndrome following an anaphylactic or anaphylactoid reaction is known as Kounis syndrome. Previous reports of Kounis syndrome described an acute coronary syndrome due either to vasospasm or atherosclerotic rupture of a coronary artery in the presence of cutaneous manifestation from the anaphylactic reaction. We report a case of a 33-year-old man who presented with anterior wall acute myocardial infarction immediately after honeybee stings without the presence of cutaneous manifestations other than the bee sting lesions. Emergent coronary arteriography revealed dissection of the proximal left anterior descending artery, which was treated with balloon dilation with an excellent outcome.

KEYWORDS Acute coronary artery dissection; acute myocardial infarction; honeybee; Kounis syndrome

Honeybee stings are common in tropical and equatorial regions of the world.[1] After the bee sting, systemic symptoms can appear rapidly and even progress to life-threatening cardiopulmonary collapse. Kounis syndrome is defined as the occurrence of an acute coronary syndrome (ACS) following an anaphylactic or anaphylactoid reaction.[2] Bee stings as the trigger of Kounis syndrome have rarely been reported. Previous reports of Kounis syndrome triggered by honeybee stings in adults included cases in which the ACS was due to either vasospasm or atherosclerotic plaque rupture. We report the first case of acute coronary artery dissection as the cause of a myocardial infarction following bee stings.

CASE PRESENTATION

A 33-year-old man presented to the emergency department at Monteria, Colombia, complaining of chest pain radiating to his back. It started immediately after multiple bee stings 14 hours earlier. During the first medical assessment, he was conscious; vital signs included blood pressure of 120/70 mm Hg, a regular heart rate of 110 beats/min, and a respiratory rate of 22 breaths/min. On the skin evaluation, he had erythematous papules on upper and lower limbs consistent with bee stings. The patient had no predisposing cardiac risk factors, such as cigarette smoking, hypertension, or diabetes. His electrocardiogram showed ST elevation in anterior and lateral leads (aVL V2, V3, V4) *(Figure 1)* and his troponin I was elevated (2.4 ng/mL; upper limit of normal: 0.04 ng/mL). Transthoracic echocardiography demonstrated hypokinesia of lateral and septal segments of the left ventricle and an ejection fraction of 55%. In the emergency department, the patient was started on medical therapy with enoxaparin 1 mg/kg, aspirin 325 mg, clopidogrel 600 mg, and atorvastatin 80 mg. Coronary arteriography revealed a dissection of the proximal left anterior descending artery *(Figure 2)*; percutaneous coronary intervention with balloon dilation was executed without any complications. The patient was asymptomatic after cardiac catheterization, and the cardiac enzymes decreased to normal levels. The patient was discharged after 4 days on aspirin 81 mg daily. The patient was followed in the cardiology clinic 1 month after discharge. He did not have any complaints, and it was decided to continue medical

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The patient agreed to use her case as a case report and allowed us to share her medical history, laboratory results, and images.

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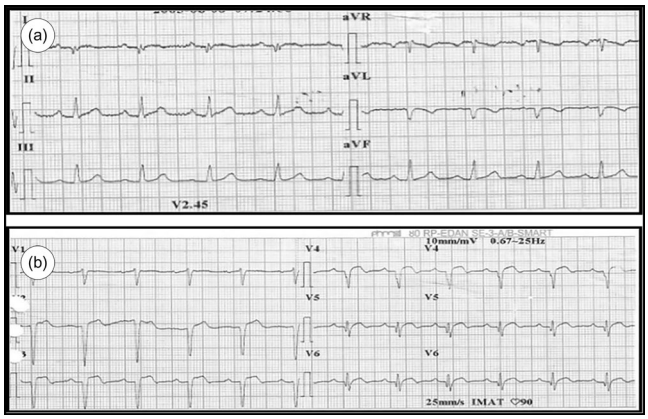

Figure 1. Electrocardiogram at the emergency department admission.

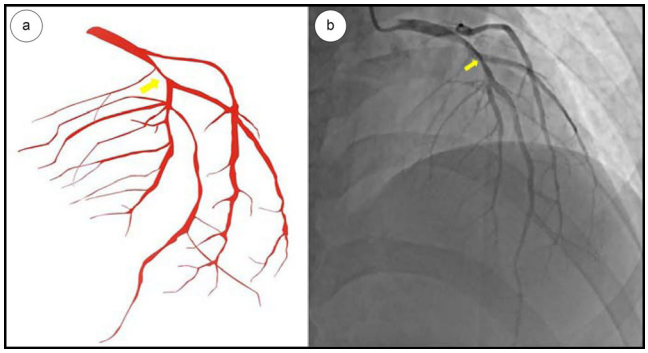

Figure 2. Coronary arteriogram. **(a)** Illustrative representation of the left coronary artery showing an acute coronary artery dissection in the proximal anterior descending artery (arrow in yellow). **(b)** Coronary arteriography of the left coronary artery showing an acute coronary artery dissection in the proximal anterior descending artery. The yellow arrow shows the location of the long, diffuse, and smooth narrowing in the artery.

management only with aspirin as an outpatient. He denied any exposure to bee stings during that month.

DISCUSSION

Honeybee venom contains a mixture of polypeptides, including mast cell degranulating peptide and melittin, that can erode coronary vessels and have been associated with cardiac toxicity and hypersensitivity.[3,4] In 1991, Kounis and Zavras[5] described a syndrome in which there was a simultaneous appearance of acute coronary events and anaphylactic or anaphylactoid reactions. The exact pathophysiologic mechanism remains unclear. Our current understanding is that exposure to an allergic stimulus activates mast cells and then eosinophils and lymphocytes, causing an injury to the walls of the coronary arteries.[2] The diagnosis of Kounis syndrome is based on medical history and physical examination.

Previous reports of Kounis syndrome triggered by honeybee stings included cases in which the ACS was due to either vasospasm or atherosclerotic plaque rupture.[6,7] Based on our literature review, this is the first case report of an ACS due to an acute coronary artery dissection of the proximal descending artery in the presence of multiple bee stings.

The patient did not have risk factors or evidence of any condition that can lead to a myocardial infarction, and even in the absence of skin manifestation, the most likely pathophysiologic explanation was Kounis syndrome. Based on previous reports, the absence of skin involvement in severe anaphylaxis has been attributed to reduced cardiac output from leakage of plasma and volume loss, which reduces venous return and prevents or delays the released anaphylactic mediators from reaching and acting on the skin.[6–8]

Kounis syndrome is likely an underdiagnosed disorder that can be secondary to coronary vasospasm, rupture of an intracoronary atherosclerotic plaque, or coronary dissection. It should be in the differential diagnosis in young, healthy patients with no atherosclerotic risk factors when they develop ACS or acute coronary artery dissection, especially after exposure to a potentially allergic agent.

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